

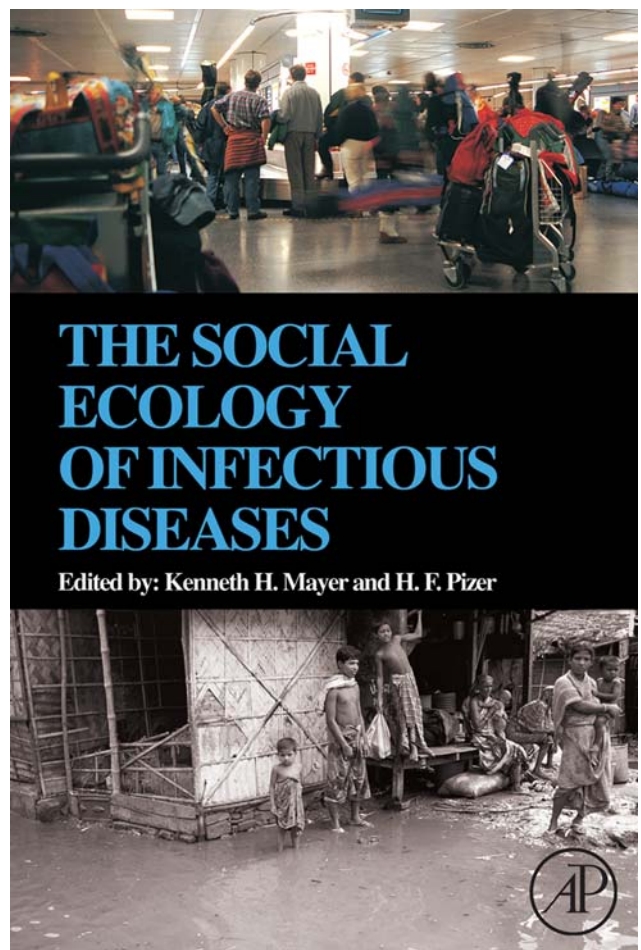
*Book Review*

## Human Infectious Diseases Through the Lens of Social Ecology

*THE SOCIAL ECOLOGY OF INFECTIOUS DISEASES*,  
KENNETH H. MAYER AND H.F. PIZER,  
(EDITORS), 2008, LONDON, UK: ACADEMIC  
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In 1787, a traveler voyaging by sailing vessel from England to Australia could expect the voyage to take about a year. Virulent infections like measles rarely reached the destination port on such trips because the length of the trip itself guaranteed that passengers were either dead or immune upon arrival. In contrast, in the 21st century, pathogens can be transported from almost anywhere on earth to almost anywhere else in less than 24 hours, a length of time shorter than the incubation period of most infectious diseases. This comparison, by authors Mary Wilson and Lin Chen, made in the second chapter of *The Social Ecology of Infectious Diseases*, nicely captures the central theme of the book. As the book's editors, Kenneth H. Mayer and H.F. Pizer, argue in their introduction, human behaviors can have a strong effect on pathogen transmission. The chapters in their book illustrate a diversity of ways in which this can happen, and how these effects have accelerated in recent decades.

The early chapters in the book describe the ways in which individual behaviors, often influenced or aided by technology, have affected disease transmission. Chapters cover the effects of travel, sexual behaviors, drug use, urbanization and suburbanization, daycare centers, blood collection, and food safety on the transmission of human pathogens. Conceptually, little in these chapters will surprise readers familiar with the classic epidemiological



models of Anderson and May. As the various chapter authors reveal in a variety of interesting examples, the number of people infected with particular human pathogens is influenced by human density, the rate of transmission between susceptible and infected people, and the rate at

which new pathogens are introduced to susceptible populations. The most interesting parts are in the details.

The chapter by Robert Pass on the transmission of cytomegalovirus (CMV) in daycare centers is particularly fascinating. CMV is an unusual pathogen in several ways: Infection with CMV rarely causes visible symptoms, viral shedding can occur for long periods of time, and transmission from mothers to offspring is possible and can lead to serious illness in infants. Pass describes how daycare centers, by facilitating horizontal transmission among children and then to their family members, have increased the transmission of CMV to previously unexposed pregnant mothers, thereby putting them at risk of transmitting CMV disease to their infants. That daycare centers are superspreading locations for pathogens will come as no surprise to readers like us who are parents of young children, but the details of CMV infection are frightening, important, and illuminating.

Superspreaders emerge as an important theme in the early chapters in the book. For example, Wilson and Chen argue that in some instances, there is covariation between the probability of acquiring infections and the probability of transmitting them. Page and Pizer provide another example in their chapter on blood safety. They argue that that blood safety is best ensured when blood must be donated, rather than sold, presumably because some of those at highest risk of transmitting blood-borne pathogens might be disproportionately likely to give blood if they were paid.

Chapters in the second half of the book emphasize distinctly human aspects of infectious disease transmission. Two interesting chapters focus on how institutions designed to combat disease have both triumphed and failed. Lubelcheck and Weinstein document the rise of antimicrobial therapies in the mid-20th century and the consequent rise in antibiotic-resistant bacteria, a problem exacerbated by physicians who frequently prescribe antibacterial agents for obviously viral infections. They also describe how social factors, including some shockingly low rates of hand hygiene by hospital workers, have driven increases in nosocomial infections. Lynch and Marcuse outline the history of vaccines and immunization, emphasizing the tremendously positive impact vaccination has had on public health while also describing the challenges for vaccine development in the 21st century, notably public perceptions of risks of immunization and economic disincentives for vaccine development.

Two chapters outline the horrendous, and not always obvious, impacts on health of two of the most distressing of human behaviors—wars and bioterrorism. These are fol-

lowed by chapters on how disasters, including anthropogenically caused climate change, influence health. Finally, the volume concludes with a series of chapters on the relationships between governance, including training and mitigation by international organizations, and infectious disease epidemics in the 21st century.

As editors of a recent book (Ostfeld et al. 2008) that focused primarily on how natural and human-dominated ecosystems both influence and are influenced by infectious disease transmission, we were struck by the similarities in some of the concepts in the two books. For example, superspreaders are well-known in diseases transmitted among wild mice, rabbits, and several other nonhuman mammals, although the underlying immunological and behavioral determinants of superspreading are poorly known. Similarly, the nonhuman equivalents of daycare centers appear to be small habitat patches, or water bodies subjected to heavy inputs of pollutants, where the animals most likely to shed pathogens aggregate and transmission rates soar. Climate change affects not only human infectious diseases; it also can exacerbate diseases of wildlife, domesticated animals, and plants.

This book and ours have differences, too. While the chapters in Mayer and Pizer's volume focus on human behaviors, the chapters in ours focus on interactions among organisms, including humans, in complex ecological systems. Ours focuses, too, on how diseases can influence the environment, as well as on how the environment can influence diseases. Comparing the two books also reveals the potential of creating a synthesis of ecology and public health, an effort already underway from both sides, as demonstrated by Mayer and Pizer's authors. Researchers interested in behavioral and institutional factors that can influence the transmission of human infectious diseases have much to learn from Mayer and Pizer's book; so, too, do ecologists who are interested in the complexities of human disease transmission.

Felicia Keesing

Biology Department

Bard College, P.O. Box 5000, Annandale-on-Hudson, NY 12583

e-mail: keesing@bard.edu

Richard S. Ostfeld

Cary Institute of Ecosystem Studies, Box AB, Millbrook, New York, NY 12545

e-mail: rostfeld@caryinstitute.org

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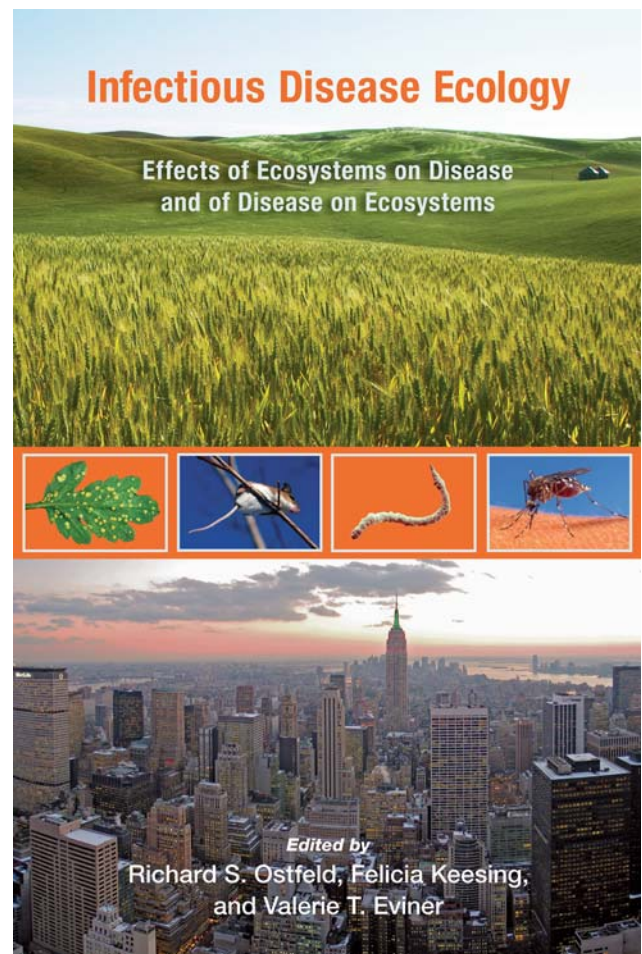
Book Review

## Ecological Studies of Diseases: Promise and Praxis

*INFECTIOUS DISEASE ECOLOGY: THE EFFECTS OF ECOSYSTEMS ON DISEASE AND OF DISEASE ON ECOSYSTEMS*, RICHARD S. OSTFELD, FELICIA KESSING, AND VALERIE T. EVINER (EDITORS), 2008, PRINCETON, NJ: PRINCETON UNIVERSITY PRESS, 504 PP, ISBN13: 978-0-691-12484-1 (CLOTH), ISBN13: 978-0-691-12485-8 (PAPER)

In the late 1960s, a number of prominent public health experts predicted the end of infectious diseases as a serious threat to mankind. Their optimism was based on successes from recently developed antimicrobial drugs, vaccines, and with infection control measures like improved public sanitation and regulating the food supply. Unfortunately, their hopeful forecast was short-lived. Soon there were epidemics caused by newly identified human pathogens, like *Legionella* and Ebola virus, and resurgent old ones like tuberculosis and malaria, often in forms resistant to previously effective antimicrobial drugs. The most catastrophic pathogen has been HIV-1, the major human immunodeficiency virus that causes AIDS. In approximately 25 years, it has spread throughout the world to infect more than 70 million people and cause approximately 35 million deaths. AIDS is now the fourth leading cause of death worldwide and accounts for about 25% of recent deaths in Africa (Kanki and Essex 2004).

We have been asked to review the Ostfeld et al. (2006) text, *Infectious Disease Ecology: The Effects of Ecosystems on Disease and of Disease on Ecosystems*, through the lens of our recently published multi-authored text, *The Social Ecology of Infectious Disease* (Mayer and Pizer, 2007). The primary unifying thesis of our book is that many, if not most, human epidemics are primarily potentiated by patterns and changes in human behavior, e.g., the sexual



revolution facilitating the spread of HIV, or increased ease of international travel leading to major global influenza epidemics and the spread of SARS. The Ostfeld et al. (2006) text is grounded in basic biological ecology, focusing on physical environment-host-pathogen interactions, while the foundations of our perspectives are public health and sociobehavioral sciences, relying heavily on medical epi-

demiology as a core investigative tool. While a superficial read of the two texts might lead one to think they are completely different, they are in fact complementary. Both texts are looking for the fundamental factors in infectious diseases that impact the health of populations, and both seek to describe and explain the complex and active interplay of pathogens and hosts that occur in an ever-changing pattern of exposures to new hosts, pathogens, and vectors, and changing physical environments.

*Infectious Disease Ecology: The Effects of Ecosystems on Disease and of Disease on Ecosystems* is the product of the 11th biennial Cary Conference that convened in May 2005 to discuss the effects of ecosystems on infectious disease dynamics. Sixty-seven scientists from diverse backgrounds who are grounded in the modern discipline of ecology contributed to the book. It is divided into four parts that explore the effects of ecosystems on disease and disease on ecosystems, management and applications, and a final chapter by the editors that looks at the challenges that lay ahead. In contrast, our book (Mayer and Pizer, 2007) is organized along the lines of the specific varied human activities that provide the most distinctive niches for microbes to flourish and spread.

One example from the two texts can help shed light on how these two books and their respective disciplines of ecology and public health inform each other. More than 75% of emerging human pathogens are zoonotic, i.e., transmitted to humans from other animals usually by vectors like mosquitoes and ticks. A myriad of human and animal activities (e.g., moving into new physical niches), plus factors like land and water use, and climate change affect the distribution and dispersal of vector populations which, in turn, influences pathogen prevalence and transmission, and ultimately infectious disease outbreaks in specific human populations. Lyme disease provides a good example of how this works. The causative agent is *Borrelia burgdorferi*, a spirochete that is typically transmitted to people by the bite of *Ixodes* ticks. While reports of *B. burgdorferi* infection in New England date back to at least the 1890s, it was not until the 1970s that Lyme disease became recognized as a significant public health problem. Michael Begon (Ostfeld et al., 2006) uses Lyme disease to explore the question of whether having multiple hosts for a given pathogen will amplify or inhibit the potential spread of an infectious disease to humans. A variety of mammals and birds that live in the northeastern United States are known to serve as reservoirs for spreading *B. burgdorferi* to humans.

Based on ecological studies of the incidence of Lyme disease in recent years, including a series of manipulative experiments with mammals, it appears that the risk of dissemination of this zoonotic infection to humans has been greater when there is relatively less diversity among potential host reservoirs. The full picture of which factors are most responsible for periods of increased clinical cases is quite complex, and many other ecological issues impact the intensity of Lyme outbreaks in humans. Examples include boom years in acorn production which can increase the number of deer reservoirs and thereby the number of ticks, and human behaviors such as the frequency of using tick repellent when working or playing outdoors in endemic areas.

In our book (Mayer and Pizer 2007), infectious disease specialist, Gary Wormser, and vector researchers, Richard Falco and Thomas Daniels, discuss how suburbanizing the landscape worked to create a favorable environment for Lyme disease. From the 1950s onward, the suburbs grew 30% faster than the cities they surround. Farmland was rapidly replaced by clustered housing separated by parks and wooded areas. While the net amount of forest did not decrease significantly after World War II, the suburbanization created a new landscape of clustered homes separated by patches of wooded land. The ideal house in the suburbs has lawn in front and back for children and pets to play, and is situated at the end of a quiet cul-de-sac street that is surrounded by conservation land. While no doubt idyllic for young families, the suburban landscape design also put people, mammals, birds, and ticks in close proximity, thereby exposing them and their pets to the bite of ticks infected with *B. burgdorferi*. Without an effective vaccine, prevention so far has depended largely on public health education around personal protection activities and behavior modification. This means using repellent when outside and promptly removing ticks from people and pets. Despite public education efforts, the number of new cases of Lyme has been increasing steadily. It just is not practical to keep children and pets indoors during the summer, or always send them outside well-covered from head to toe and sprayed with DEET insecticide. Meanwhile, most suburban communities have been against public prevention strategies that could be aimed at controlling vectors and reservoir hosts, like widespread pesticide spraying and culling deer populations. Research shows that Lyme disease is driven largely by the abundance of nymphal *I. scapularis* (Ostfeld et al. 2006). Looking ahead, it would seem that strategies for controlling Lyme outbreaks are likely to be

informed by ecological studies such as those described by Begon in *Infectious Disease Ecology*.

There are other examples in this text of how basic ecology helps inform the public health approach to infectious disease outbreaks in humans. In Chapter 4, Johnson and Carpenter discuss consequences of eutrophication, which occurs when excessive nutrients, like nitrogen and phosphorous from sewage and fertilizer run off, produce plant overgrowth and decay. The algal blooms that result can adversely impact water quality and marine life, as well as create opportune environments for microbial pathogens to flourish that can cause human disease. Eutrophication has been associated with outbreaks of a type of allergic dermatitis (“swimmer’s itch”) that occurs when trematode cercariae invades the skin, as well as cholera and malaria. In addition to eutrophication, land clearing produces large numbers of temporary pools for mosquitoes to breed and spread malaria. On the other hand, if eutrophication is not abetted by other human influences, such as overwhelmed sewerage systems in developing world slums in mega-cities, or poor sanitation in public bathing areas, its sequelae may not result in appreciable clinical and public health problems.

To the modern ecologist, infectious disease outbreaks are inherent events that inevitably will occur and be structured by specific ecosystem dynamics. Basic research is needed to understand how and why pathogenic organisms, and the reservoirs and vectors associated with their spread to humans, are able to thrive in different environments. Although ecology is a theoretical discipline that does not

focus principally on predicting the next emerging or reemerging pathogen or how it can be controlled, it is increasingly being applied to these questions and, over time, it will likely play a more important role in infectious disease control and public health planning.

Kenneth H. Mayer

Brown University Medical School, Providence, RI

Kenneth H. Mayer

Brown University, 1313 Washington Street, Apt. 713,  
Boston, MA 02118-2171

e-mail: [Kenneth\\_Mayer@Brown.edu](mailto:Kenneth_Mayer@Brown.edu)

H. F. Pizer

Health Care Strategies, Inc, Cambridge, MA

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